Tumor Lysis Syndrome

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KEYWORDS

• Tumor lysis syndrome • Acute kidney injury • Rasburicase • Hyperuricemia
• Oncologic emergency

KEY POINTS

- Tumor lysis syndrome is a potentially life-threatening oncologic emergency resulting in severe electrolyte derangements that may lead to kidney failure, seizures, arrhythmias, and death.
- Clinicians should have a high index of suspicion for tumor lysis syndrome, particularly in patients with high-risk malignancies, pre-existing kidney disease, or large tumor burden.
- Treatment includes rigorous hydration and antihyperuricemic medications, such as rasburicase.
- Prevention of tumor lysis syndrome is the most important intervention for intermediate-risk to high-risk patients.

INTRODUCTION

Tumor lysis syndrome (TLS) is an oncologic emergency characterized by the massive destruction of tumor cells and the release of intracellular components into systemic circulation. The subsequent development of hyperkalemia, hyperuricemia, hyperphosphatemia, and hypocalcemia results in significant patient pathology including acute kidney failure, seizures, arrhythmias, and potentially sudden death. TLS typically occurs in patients with hematologic malignancies following treatment with cytotoxic chemotherapy, though it may also occur spontaneously in patients with a high tumor burden. With the increase in the number of patients who are diagnosed with cancer and the use of more aggressive cancer treatment therapies, the incidence of TLS is increasing. It is, therefore, imperative that physicians are able to efficiently recognize, diagnose, and treat this life-threatening condition.

CLINICAL CASE

A 38 year old female patient with a history of acute myeloid leukemia (AML) presents to the emergency department (ED) with nausea, vomiting, and fatigue. The patient

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Emerg Med Clin N Am 43 (2025) 453–461
https://doi.org/10.1016/j.emc.2025.04.002 **emed.theclinics.com**
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Abbreviations	
ACE	angiotensin converting enzyme
AKI	acute kidney injury
AML	acute myeloid leukemia
CTLS	clinical TLS
ECG	electrocardiogram
ED	emergency department
GI	gastrointestinal
HD	hemodialysis
ICU	intensive care unit
IV	intravenous
LDH	lactate dehydrogenase
LTLS	laboratory identified TLS
TLS	tumor lysis syndrome
WBC	white blood cell

received her first round of chemotherapy 2 days ago and soon after began to feel unwell. She endorses dizziness, generalized weakness, myalgias, nausea, vomiting, and reduced urine output. She denies fever, cough, congestion, shortness of breath, chest pain, or dysuria.

On examination, the patient is tachycardic with a heart rate of 124, mildly tachypneic with a respiratory rate of 22, and afebrile. She is awake but lethargic and confused with dry mucus membranes. She has normal heart and breath sounds. Her abdomen is soft but diffusely tender without guarding.

Significant Results

White blood cell count (WBC) 103,000/μL; hemoglobin 7.9 g/dL; and platelets 45,000/μL

K 6.4 mmol/L; BUN 49 mg/dL; creatinine 4.1 mg/dL; calcium 6.8 mg/dL; and uric acid 11.9 mg/dL

Phosphate 5.9 mg/dL

Lactate dehydrogenase (LDH) 950 U/L

Lactate 4.5 mmol/L

Electrocardiogram (ECG) is notable for sinus tachycardia, otherwise within normal limits

The patient is emergently started on intravenous (IV) fluids at 250 mL/h and rasburicase is ordered for treatment of hyperuricemia. Ondansetron is administered to improve her nausea and vomiting. Given the absence of ECG changes, calcium is avoided to prevent worsening of her renal function; insulin with dextrose and high-dose albuterol nebulization are given to treat the hyperkalemia. Nephrology is consulted for assistance with her acute kidney injury (AKI) and electrolyte derangement. The patient is admitted to the intensive care unit (ICU) for continued management of her TLS with IV hydration, rasburicase, frequent laboratory monitoring, and oncology consultation.

DISCUSSION

Etiology and Epidemiology

TLS most often occurs in patients with hematologic malignancies such as Burkitt's lymphoma, acute lymphocytic leukemia, and AML.[1,2] Though less common, the incidence of solid tumors causing TLS has increased; hepatocellular carcinoma, lung cancer, melanoma, breast cancer, neuroblastoma, and germ cell tumors are associated with an elevated risk for the development of TLS.[2–4]

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The incidence of TLS is variable. One study found that in treatment-related TLS, the overall incidence was 9.3% for laboratory identified TLS (LTLS, using the Cairo–Bishop definition) and 6.7% for clinical TLS (CTLS, defined as laboratory TLS plus one clinical complication, also using the Cairo–Bishop definition, **Table 1**). This same study noted that rates were highest for hematologic cancers (21.1% LTLS and 15.1% CTLS) followed by gastrointestinal (GI; 8.4% LTLS and 5.0% CTLS) and then epithelial (4.9% LTLS and 4.0% CTLS) malignancies. They identified acute leukemia and multiple myeloma as the most prevalent hematologic cancers and esophageal as the most common solid tumor causing TLS.[5]

Another study found that between 2016 and 2019, the incidence of TLS was 36 per 100,000, or 0.036%. Additionally, TLS is diagnosed more commonly in male patients.[6] Given the incredibly variable prevalence of this oncologic emergency, it is unclear what the actual incidence of TLS may be.

Pathophysiology

TLS typically occurs within 72 hours of cytotoxic chemotherapy treatment.[1] It may also occur spontaneously, specifically in patients with a high tumor burden, and more rarely following radiation therapy, immunotherapy, corticosteroid administration, or hormonal therapy.[2]

Tumor cell lysis, whether following treatment or spontaneously, causes the release of electrolytes, proteins, and cellular DNA into systemic circulation. DNA, composed of nucleic acids, are then rapidly broken down by the liver into uric acid resulting in accumulation and hyperuricemia followed by its crystallization within renal tubules.[2,3] Phosphate, from the breakdown of cytosol, leads to the formation of calcium phosphate, which then precipitates within renal tubules and causes hypocalcemia. Due to the kidney's impaired ability to filter and excrete substances, potassium, from the breakdown of cytosol and arguably the most lethal component, accumulates resulting in hyperkalemia.[7]

Certain factors have been found to increase the risk of developing TLS, each of which contributes to the pathophysiology of the condition. These risk factors include dehydration, chronic kidney disease, pre-existing uremia, and elevated LDH or uric acid levels pretreatment. Additionally, malignancy-related risk factors include high WBC count, tumor burden or bulkiness, rapid tumor growth, metastasis, and tumors with high sensitivity to chemotherapy.[1,2,4,7]

Table 1
Cairo–Bishop diagnostic criteria for tumor lysis syndrome[2,3,7]

Laboratory Diagnosis	Clinical Diagnosis
At least 2 of the following criteria are met within a 24 h period, occurring 3 d before or 7 d after chemotherapy initiation:	If criteria are met for laboratory diagnosis *and* at least 1 of the following clinical criteria below is present:
Uric acid $\geq$8 mg/dL or 25% increase from baseline	Rise in serum creatinine 1.5 times the upper limit of normal
Potassium $\geq$6 mEq/L or 25% increase from baseline	Cardiac arrhythmia or sudden death
Phosphorus $\geq$4.5 mg/dL (adults) or 25% increase from baseline	Seizure
Corrected calcium $\leq$7 mg/dL or 25% decrease from baseline	

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Clinical Manifestations

Patients who present to the ED, urgent care center, or physician office should be assessed for direct tumor effects and any potential oncologic emergency. A thorough review of systems and physical examination will assist with the proper assessment and workup for each patient.

Presentation of patient with TLS can range between asymptomatic with incidental laboratory abnormalities (LTLS) to cardiac arrest and sudden death. Clinical manifestations result from the sequelae of abnormalities in potassium, uric acid, phosphate, and calcium, and subsequent organ damage. In patients with pre-existing risk factors, particularly renal insufficiency, they are more likely to present with higher severity signs and symptoms of TLS.[7]

Symptoms related to TLS will often be nonspecific. One study identified GI (33%), genitourinary (33%), and neurologic (26%) symptoms as the most frequently reported in patients who have been diagnosed with TLS.[4] Ultimately, the exact presentation will vary based on the severity of electrolyte derangements.

Hyperkalemia can cause weakness, fatigue, muscle cramps, paresthesias, palpitations, syncope, and dysrhythmias.[7,8] Depending on the severity of potassium elevation, ECG changes may include peaked T waves, increased PR interval, QRS widening, loss of the P wave, and sine wave (**Table 2**). Complete heart block, ventricular dysrhythmias, and asystole may also occur. Renal failure, metabolic acidosis, potassium-sparing diuretics, and other medications that may increase potassium levels, such as angiotensin converting enzyme (ACE) inhibitors can worsen hyperkalemia in patients with TLS.[7]

The kidneys excrete uric acid from the body through the proximal renal tubules. When uric acid levels accumulate due to massive cell lysis, renal tubule transporters become saturated resulting in precipitation of uric acid crystals, urate nephropathy, and AKI. Hyperuricemia is exacerbated by pre-existing renal failure, dehydration, conditions that may result in dehydration such as vomiting and diarrhea, and diabetes insipidus. Additionally, the development of uremia and obstructive uropathy leads to low urine flow rates further worsening hyperuricemia.[7] Patients may, therefore, present with generalized weakness, fatigue, malaise, nausea, vomiting, pruritus, joint pain, and/or signs and symptoms of fluid overload.[2]

Compared to normal cells, malignant cells can contain up to 4 times the amount of phosphorus resulting in hyperphosphatemia with the rapid destruction of tumor cells, a phenomenon that typically occurs within 24 to 48 hours of the initiation of chemotherapy. As with hyperuricemia, the kidneys become saturated and unable to excrete this molecule resulting in the precipitation of calcium phosphate in renal tubules, further exacerbating the AKI seen in TLS.[7] In most cases, patients with hyperphosphatemia are

Table 2
Electrocardiogram changes that may be seen with hyperkalemia and hypocalcemia[7–9]

Electrolyte Abnormality	ECG Changes
Hyperkalemia	Peaked T waves Wide QRS Loss of P wave Sine wave
Hypocalcemia	QT prolongation Prolonged ST segment T wave flattening or inversion ST elevation

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often asymptomatic.[10] However, patients may develop GI symptoms, such as nausea, vomiting, and diarrhea, or lethargy and seizures.[7] More often, patients will exhibit symptoms of hypocalcemia, which may include Chvostek sign, Trousseau sign, muscle cramps, carpal or pedal spasm, tetany, altered mental status, seizures, delirium, or hypotension.[2,7,10] Hypocalcemia may also produce ECG changes (see **Table 2**). Most commonly, patients may experience a prolonged QT interval, proportional to the degree of hypocalcemia, due to the elongation of the ST segment. T wave flattening or inversion, exacerbation of arrhythmia from other etiologies, or, more rarely, ST elevation can occur, mimicking acute myocardial infarction.[7,9]

Diagnosis

The Cairo–Bishop criteria is the most widely used diagnostic criteria for TLS, initially set forth in 2004, but later revised by Howard and colleagues in 2011. Laboratory (LTLS) diagnosis and clinical (CTLS) diagnosis of TLS are depicted in **Table 1**.[11] LTLS diagnosis can be made if 2 or more of the following criteria are present within a 24 hour period:

- Uric acid 8 mg/dL or greater or 25% increase from baseline
- Potassium 6 mEq/L or greater or 25% increase from baseline
- Phosphorus 4.5 mg/dL or greater (in adults) or 25% increase from baseline
- Corrected calcium 7 mg/dL or less or 25% decrease from baseline

CTLS is diagnosed when criteria are met for LTLS and at least one of the following clinical criteria are present: rise in serum creatinine 1.5 times the upper limit of normal for age and gender, seizure, and/or cardiac arrhythmia or sudden death. It is important to know that the Cairo–Bishop criteria specifically identify TLS as it is related to chemotherapy; however, TLS can also occur spontaneously. Additionally, the creatinine criteria do not account for patients who may have underlying chronic kidney disease.[2,7,11]

Management

Patients who have been diagnosed with TLS require rapid treatment to prevent further worsening of their condition and subsequent death. Aggressive hydration and uric acid-lowering agents are the mainstays of treatment of TLS. Other therapies are dependent on severity of the patient's condition, electrolyte abnormalities, and clinical signs and symptoms.

Rigorous hydration is indicated for all patients who have been diagnosed with TLS to improve glomerular filtration, renal perfusion, and impart a high urine output. IV crystalloid choice is dependent on the patient's electrolyte abnormalities and clinical status. In general, patients should initially receive isotonic saline if they are hypovolemic or hyponatremic.[12] It is recommended that patients receive approximately 2 to 3 L of IV fluid per day for both adults and children; children who weigh 10 kg or less should receive 200 mL/kg/d. Urine output goal is 80 to 100 mL/h or 4 to 6 mL/kg/h in patients weighing less than 10 kg. Use caution in patients who have a history of chronic kidney disease, liver failure, or congestive heart failure as aggressive hydration can result in fluid overload and respiratory failure. These patients, or any patient with evidence of fluid overload, can receive concomitant diuresis to aid in electrolyte excretion and prevention of further fluid retention. For patients who receive concomitant diuretics, potassium-lowering loop diuretics are preferred, such as furosemide.[7] IV hydration should continue until LDH has normalized.[12]

Medications to lower uric acid levels (referred to as antihyperuricemic agents or hypouricemic therapy) should be considered in all patients with TLS. Rasburicase,

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recombinant urate oxidase, is administered to enhance the elimination of uric acid. Urate oxidase converts uric acid into allantoin, which is 5 to 10 times more soluble in urine than uric acid and, therefore, easily excreted by the kidneys. Rasburicase acts to decrease existing uric acid through the conversion to allantoin; it does not prevent the formation of new uric acid.[3,7,12] Rasburicase acts rapidly, within hours, and has few side effects. It is contraindicated in patients with glucose-6-phosphate dehydrogenase deficiency and in those who are pregnant or lactating. The recommended dose of rasburicase is 0.2 mg/kg/day for 5 days, though length of treatment is dependent on responsiveness to the medication and clinical improvement. Some studies have noted that rasburicase may be effective at much smaller doses and given the financial cost of the medication, it is worthwhile to determine the need for further dosing on a per patient basis.[7,12]

Allopurinol inhibits the conversion of xanthine to uric acid, thereby inhibiting its formation. Allopurinol prevents the production of new uric acid but does not treat hyperuricemia as rasburicase does. Allopurinol can be used in the treatment of TLS at a maximum dose of 800 mg/day divided every 8-12 hours. In patients with renal insufficiency, dosing should be reduced. Allopurinol increases the level of xanthine, which can lead to xanthine nephropathy potentially worsening the obstructive nephropathy seen in TLS. Some advocate to avoid the use of allopurinol in patients with confirmed TLS.[2,7,12] Side effects of the medication include rash, and it should be used cautiously in patients taking azathioprine and other immunosuppressive drugs.[2,12]

Febuxostat is a new antihyperuricemic agent that is also a xanthine oxidase inhibitor that can be used in patients who are allergic to or cannot tolerate allopurinol. Febuxostat has fewer drug interactions and less potential impact on renal function; however, it is more expensive than allopurinol and evidence does not demonstrate similar effectiveness.[2,12]

Hyperkalemia is the most lethal consequence of TLS due to its propensity to cause life-threatening arrhythmias resulting in cardiac arrest and death. Hyperkalemia secondary to TLS should be treated similarly to other etiologies of this electrolyte derangement. Patients require frequent monitoring of their potassium levels, typically every 4 to 6 hours, and continuous telemetry monitoring.[7] Depending on the severity of hyperkalemia and development of ECG changes, patients may require IV calcium administration, potassium-lowering agents (sodium zirconium cyclosilicate, furosemide, and so forth), temporizing medications with IV insulin-dextrose and inhaled albuterol nebulization, or hemodialysis.

Hyperphosphatemia should be treated with IV fluid hydration and oral phosphate binders to decrease intestinal absorption of phosphate. For severe or refractory hyperphosphatemia, dialysis may be indicated. Hyperphosphatemia should be treated prior to the treatment of hypocalcemia in patients with TLS as exogenous administration of calcium medications can result in further precipitation of calcium phosphate crystals in renal tubules exacerbating AKI.[2,7] However, for patients who are symptomatic from hypocalcemia with ECG changes and/or arrhythmias, calcium replacement therapy should be initiated immediately. Magnesium levels should also be monitored and repleted if found to be low.[1]

Hemodialysis (HD) should be considered in patients with life-threatening electrolyte derangements or significant loss of kidney function. Specific criteria include persistent hyperkalemia greater than 6 mEq/L, severe hyperuricemia greater than 10 mg/dL or renal failure (creatinine >10 mg/dL), symptomatic hypocalcemia with a serum phosphate level greater than 3.3 mmol/L, and oliguria or anuria[1] as well as typical indications for emergent HD such as volume overload, respiratory failure secondary to pulmonary edema, and uremia with altered mental status. Patients with TLS often

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have excellent renal recovery with treatment, warranting a low threshold for initiation of HD or continuous hemofiltration.[7]

Alkalization of the urine was previously a common treatment of TLS in an effort to increase the solubility and, therefore, excretion of uric acid. However, it has been shown that this may increase the precipitation of calcium phosphate crystals and further worsen hypocalcemia and kidney failure in patients with TLS.[7] Urinary alkalinization should only be used in patients who have severe hyperuricemia when rasburicase is not available.[1,7]

Disposition

Patients who are diagnosed with TLS should be admitted to the ICU for continuous telemetry and close monitoring. Serum electrolytes, creatinine, and uric acid should be monitored every 4 to 6 hours. Rasburicase and IV fluid hydration should be continued until uric acid and LDH levels have normalized. Strict intake and output volumes should be monitored. For patients who have worsening renal function, refractory volume overload, or worsening electrolyte abnormalities, consider continuous renal replacement therapy.[11,12]

Patients may present to the clinic or the ED with signs of early TLS. While their laboratory abnormalities may not be consistent with LTLS, it is imperative that they receive prophylactic inpatient therapy to prevent further worsening of their condition. Early TLS is evident in patients with intermediate-risk to high-risk factors or signs of TLS with one or less laboratory abnormality, as defined by Cairo–Bishop. These patients are classified as "at risk" and should be admitted for IV fluid hydration, monitoring of electrolytes and renal function every 8 to 12 hours, and consideration for rasburicase or allopurinol.[7]

Prevention

The most important intervention in the management of TLS is to prevent TLS from occurring. Preventative measures can be implemented prior to initiation of chemotherapy, particularly in intermediate-risk or high-risk patients. Preventative measures include monitoring hydration and utilizing hypouricemic therapy.

Patients must be risk stratified at the time of oncologic diagnosis and prior to the initiation of cytotoxic chemotherapy. Puri and colleagues[12] propose that the risk factor assessment should include malignancies that have been identified previously as high risk for the development of TLS as well as tumor-related factors (eg, high proliferation rate, chemosensitivity, and so forth), and patient-centered factors (eg, pretreatment hyperuricemia, chronic kidney disease, and so forth). For patients who are found to be intermediate or high risk for TLS, preventative monitoring and therapy should be initiated.

Patients who are identified as being at risk should have IV fluid hydration started 1 to 2 days prior to the initiation of chemotherapy and continued for 2 to 3 days following the completion of the cytotoxic therapy.[2,7] Additionally, patients should have their electrolytes, LDH levels, creatinine, and uric acid levels monitored.[1] Allopurinal should be started at least 24 hours prior to the start of cytotoxic therapy and continued until uric acid levels have normalized and/or evidence of tumor burden has lessened.[13] Medications that can vasoconstrict renal vasculature, such as iodinated contrast or nonsteroidal anti-inflammatory drugs, or increase potassium, such as ACE-inhibitors or spironolactone, should be avoided.[2,7]

SUMMARY

TLS is a well-known oncologic emergency. Massive cell lysis that often occurs following the initiation of cytotoxic chemotherapy results in the release of cellular contents

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causing patients to develop severe hyperuricemia, hyperkalemia, hyperphosphatemia, hypocalcemia, and acute renal failure. Patients will present with symptoms related to these underlying metabolic derangements, potentially with life-threatening sequelae of seizures, arrhythmias, and cardiac arrest. TLS necessitates emergent and rapid treatment with IV fluid hydration, rasburicase, and supportive care to improve the electrolyte abnormalities and loss of kidney function seen in this patient population. The most important step in the management of TLS is to prevent the illness from occurring with prophylactic IV fluids, allopurinol, and frequent electrolyte, creatinine, and LDH monitoring. TLS carries a high risk for morbidity and mortality; however, our ability to prevent and treat this condition is improving, resulting in a less grim prognosis overall.

CLINICS CARE POINTS

- TLS is an oncologic emergency that requires emergent management and coordinated care by oncology, nephrology, and intensive care clinicians.
- Providers must maintain a high index of suspicion for the prevention and diagnosis of TLS. At-risk patients, such as those with hematologic malignancies, high proliferation rate, large tumor burden, and pre-existing kidney failure, should be closely monitored and treated prophylactically.
- Hyperkalemia is the most lethal metabolic derangement of TLS and necessitates rapid therapy based on the potassium level and ECG changes.
- Patients with TLS should be treated with aggressive IV fluid hydration and antihyperuricemic agents, such as rasburicase.
- Those that have been diagnosed with TLS require admission to the ICU for continuous telemetry and monitoring of electrolytes, creatinine, and uric acid every 4 to 6 hours. Clinicians should have a low threshold for the initiation of hemodialysis in patients who are not improving despite aggressive care.

DISCLOSURE

The authors have nothing to disclose.

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